carcinomas. The principal reason for the distinction between adenomas and adenocarcinomas of the kidney on the basis of size derives from Bell's statement that "tumors under 3 cm in diameter have rarely formed metastases."[3] Admittedly, this pathologic distinction is an arbitrary one and no consonant opinion exists among pathologists. Certainly all adenocarcinomas begin as small tumors, and since microscopically adenomas and adenocarcinomas are generally indistinguishable, it is perhaps logical that distinctions are made on the basis of the stage of growth alone.

In the present case, therapy was partial nephrectomy, which, considering the size of the tumor and its pseudoencapsulation, appeared to be adequate treatment. Vermooten advocated partial nephrectomy for clear cell carcinoma.[12] Conservation of as much renal tissue as possible was strongly indicated in the present case, particularly in light of the finding of focal glomerulonephritis in the remaining kidney.

The patient and his siblings resemble those with hereditary chronic nephritis previously reported.[2,8,10] Neither the patient nor his siblings manifested extrarenal abnormalities of the kind seen in some patients with hereditary chronic nephritis—nerve deafness or abnormalities of the eyes, skin or bone. The pathologic features in this case were consistent with early forms of the disorder in showing minor nonspecific changes such as hyalinized glomeruli. Lipid-filled foam cells in the interstitium between the tubules near the corticomedullary junction were not seen. A review of the literature, however, disclosed that the histologic picture of familial nephritis is not consistent and that some patients have had typical interstitial pyelonephritis whereas others have had glomerular crescents and hypercellularity.[10]

Summary

A case of early carcinoma of the kidney was detected accidentally on needle biopsy in the study of a patient with familial nephritis. The lesion, 1.2 cm in diameter, was a solitary one in the lower pole of the kidney. The lesion was confirmed by angiography which revealed a characteristic vascular tumor pattern. Treatment was lower pole nephrectomy. Microscopically the tumor was an adenocarcinoma of the kidney, although on the basis of size alone some pathologists would classify it as an adenoma.

REFERENCES

1. Ackerman, L. B.: Surgical Pathology, 2nd ed., C. V. Mosby Co., St. Louis, 1959.

2. Alport, A. C.: Hereditary familial hemorrhagic nephritis, Brit. Med. J., 1:504, March 1927.

3. Bell, E. T.: A classification of renal tumors with observations on the frequency of the various types, J. Urol., 39:238, March 1938.

4. Björnberg, O: Hypernephrom i seniet, Nord. Med., 64:1005, 1960.

5. Böttiger, L. E.: Studies in renal carcinoma, I, Acta. Med. Scandinav., 167:443, 1960.

6. Böttiger, L. E., Hallberg, C., and v Schreeb, T.: Renal carcinoma as an accidental finding, Acta. Chir. Scandinav., 127:158-161, 1964.

7. Bunge, R. G., and Kraushaar, O. F.: An early renal malignancy, diagnosed preoperatively, J. Urol., 63:475-479, March 1950.

8. Goldman, R., and Haberfelde, G. D.: Hereditary nephritis: Report of a kindred, New Engl. J. Med., 251: 734, October 1959.

9. Kaufman, J. J., and Mims, M.: Tumors of the kidney, Curr. Prob. Surg., pp. 1-44, February 1966.

10. Perkoff, G. T., Nugent, C. A., Dolowitz, D. A., Stephens, F. E., Carnes, W. H., and Tyler, F. H.: A follow-up study of hereditary chronic nephritis, Arch. Int. Med., 102:733, November 1958.

11. Uys, C. J.: Tumours of the kidney in the Bantu races of South Africa, Brit. J. Urol., 28:75, March 1956.

12. Vermooten, V.: Indications for conservative surgery in certain renal tumors: A study based on the growth pattern of the clear cell carcinoma, J. Urol., 64: 200, August 1950.

13. Willis, R. H.: Pathology of Tumors, 3rd ed., Butterworth, London, 1960.

Hemorrhage from The Gallbladder

A Report of Three Cases

SANFORD POLSE, M.D., RONALD J. STONEY, M.D., AND JOHN N. BALDWIN, M.D.,
San Francisco

NONTRAUMATIC HEMORRHAGE from the gallbladder (hemorrhagic cholecystitis) is a rare complication of biliary tract disease. Since in signs and symptoms it may mimic several other diseases, the diagnosis is often delayed and is usually not made until operation or autopsy. The symptoms usually resemble those of acute cholecystitis, although the primary diagnosis may be massive upper gastrointestinal bleeding, hydrops of the gallbladder,

From the Departments of Surgery, University of California School of Medicine, and the San Francisco General Hospital, San Francisco.

Submitted 12 December 1966.

Reprint requests to: Department of Surgery, University of California School of Medicine, San Francisco 94122 (Dr. Baldwin).

hemoperitoneum or obstruction of the common duct.

Few cases of nontraumatic hemorrhage from the gallbladder have been reported. In 1961, Fitzpatrick[2] reviewed the literature on hemocholecysts (nontraumatic hemorrhage from the gallbladder without free intraperitoneal rupture), and collected 34 cases, while adding one of his own. In 1961 Raycroft and Mastrangelo[5] reviewed the literature on massive intraperitoneal hemorrhage as a complication of gallbladder disease; they found 16 somewhat similar cases and added two of their own. Our recent experience with three cases of hemorrhage from the gallbladder prompted a further review of the subject and the following case reports.

Reports of Cases

CASE 1.—A 58-year-old white man entered San Francisco General Hospital 1 April 1965 with a one-week history of progressive pain in the right upper quadrant of the abdomen, nausea, and vomiting of clear, yellowish material. He had no recent history of trauma, hematemesis or melena. In 1955 he was told that he had cirrhosis of the liver, and he had a bleeding peptic ulcer in 1959. The temperature was 39.0°C (102.2°F). The right upper quadrant of the abdomen was tender and a large, somewhat tense mass measuring 8 by 10 by 10 cm was palpable as well as visible. Rectal examination was normal and the stool guaiac test was negative for blood. Leukocytes numbered 5,500 per cu mm of blood and the hematocrit was 43 per cent. Prothrombin time was 64 per cent. A plain roentgenogram of the abdomen showed multiple calculi in the right upper quadrant in what appeared to be a large, distended gallbladder.[1] After conservative treatment the patient rapidly became asymptomatic and within eight hours after admission the mass was no longer palpable. However, the mass reappeared 36 hours later, and symptoms became worse. Surgical intervention was then considered mandatory. At operation the gallbladder was large, distended, edematous and inflamed. On aspiration, 750 ml of blood, dark at first and then bright red, was withdrawn. When the gallbladder was opened, the entire mucosa appeared to be bleeding. Multiple calculi were found, including an impacted cystic duct stone. Cholecystectomy was performed. During operative cholangiography, clear golden bile was obtained from a normal-appearing common duct. Postoperatively the patient was transiently icteric. On the fourth postoperative day, he passed a black tarry stool which was guaiac-positive. Gastric aspirate was guaiac-negative. Recovery then was satisfactory.

Pathologic examination of the gallbladder showed acute hemorrhagic cholecystitis. The wall was thickened and acutely inflamed in all layers. Hemorrhage and edema of the submucosa, with sloughing of the mucosa, were present.

Comment: Acute hemorrhagic cholecystitis manifested itself as rapidly recurring hydrops, prompting surgical intervention.

CASE 2.—An 82-year-old white man was admitted to the San Francisco General Hospital 15 October 1964 with a 12-hour history of upper abdominal pain, and vomiting of "coffee grounds" material. He was disoriented and a more extensive history could not be obtained. Blood pressure was 88/55 mm of mercury and the pulse was 128. The abdomen was slightly distended and a fluid wave was present. Leukocytes numbered 14,200 per cu mm of blood, and the hematocrit was 46 per cent. A plain roentgenogram of the abdomen showed no abnormality. Paracentesis yielded large quantities of bloody fluid. At laparotomy approximately 1,000 ml of bloody fluid was present in the peritoneal cavity. The gallbladder was acutely inflamed and gangrenous and had a perforation 2 cm long, which was bleeding briskly. The diffuse bleeding appeared to come from the entire mucosa. Cholelithiasis was also present. The stomach and duodenum were normal. Cholecystectomy was performed. Postoperatively, the patient did well at first but his condition gradually deteriorated and nine days after operation he died of pulmonary complications.

Comment: Acute hemorrhagic cholecystitis, an unusual cause of hemoperitoneum, was controlled by cholecystectomy.

CASE 3.—A 55-year-old white man was admitted to the San Francisco General Hospital 20 September 1965 with a three-day history of nausea, vomiting and generalized abdominal pain. He had a past history of severe claudication of the lower extremities. Aorto-iliac thromboendarterectomy had been performed in June 1965. The temperature was 38.5°C (101.3°F). The entire abdomen was rigid and pronounced guarding was evident in all quadrants. Bowel sounds were hypoactive. Leukocytes numbered 33,000 per cu mm of

blood and the hematocrit was 36 per cent. Roentgenograms of the abdomen showed dilated loops of small bowel and right colon, which contained air-fluid levels. Nasogastric aspirate and stool were positive for occult blood. The preoperative diagnosis was superior mesenteric artery occlusion. At laparotomy, no abnormality was found in the small bowel, but approximately 300 ml of dark, viscous blood was seen in the infrahepatic space. The gallbladder was greatly dilated and hemorrhagic; a recent perforation was noted in the posterior portion of the cystic ampulla. Several cholesterol stones measuring 3 to 4 mm were floating free in the pericholecystic area. Cholecystectomy was carried out. The postoperative course was uneventful. Microscopic examination of the gallbladder showed hemorrhagic material within the lumen and considerable amounts of extravasated blood dissecting through the gallbladder wall.

Comment: Acute hemorrhagic cholecystitis mimicked superior mesenteric vascular occlusion, with gastrointestinal bleeding. Hemobilia caused anemia, free-peritoneal bleeding and occult blood in stools and nasogastric aspirate.

Discussion

Bleeding, an infrequent complication of gallbladder disease, is usually caused by gallstones.[2] Acute hemorrhagic cholecystitis was associated with cholelithiasis in all three of the cases here reported. The wide variety of presenting symptoms often delays the correct diagnosis.[3,4,6] In Case 1, the rapidly reappearing hydrops might have alerted us to hemorrhage into the gallbladder. As the gallbladder initially distended, the stone in the cystic duct apparently became disimpacted and the gallbladder decompressed as blood passed into the bowel through the common duct. Later, a stone probably again became impacted and the mass reappeared. The fact that the patient passed a tarry stool on the fourth postoperative day further confirms this impression.

The combination of hemoperitoneum and peritonitis usually results from perforated and bleeding duodenal ulcers. Hemoperitoneum as a complication of gallbladder disease is usually due to erosion of the cystic artery or one of its branches, or to an area of focal necrosis at the site of perforation.[5] In Case 2 a perforation was found, but the mucosa of the gallbladder appeared to be oozing blood from its entire surface. Cholecystectomy effectively controlled the hemorrhage. The patient died later of pulmonary complications.

In Case 3, both hemoperitoneum and evidence of gastrointestinal blood loss were found. Gross bleeding had occurred into the lumen of the gallbladder, and before perforation the organ had decompressed by way of the cystic duct into the duodenum. Extravasation of blood was found in all layers of the acutely inflamed gallbladder, which was also the site of acute cholecystitis.

In other reported cases of acute hemorrhagic cholecystitis with hemobilia, gastrointestinal blood loss was severe enough to warrant exploratory laparotomy because of a presumed diagnosis of hemorrhaging duodenal ulcer.[1,3] Since the gallbladder may be a source of enteric as well as intraperitoneal blood loss, hemorrhage can usually be treated successfully by prompt operative intervention.

Summary

Three cases of hemorrhagic cholecystitis with different clinical manifestations are described. One patient had acute cholecystitis with recurrent hydrops, another had hemoperitoneum and the third had hemoperitoneum with gastrointestinal bleeding. Cholecystectomy successfully controlled the hemorrhage in all cases. The biliary tract should be considered as a possible source of bleeding in patients with obscure hemoperitoneum and upper gastrointestinal hemorrhage.

REFERENCES

1. Engler, H. S., Zavaleta, A. A., and Moretz, W. H.: Hemobilia, Amer. Surgeon, 30:756, 1964.

2. Fitzpatrick, T. J.: Hemocholecyst, Ann. Intern. Med., 55:1008, 1961.

3. Hudson, P. B., and Johnson, P. P.: Hemorrhage from the gallbladder, New Engl. J. Med., 234:438, 1946.

4. Mailer, R.: Spontaneous rupture of the gallbladder with massive intraperitoneal haemorrhage, Brit. J. Surg., 27:91, 1939.

5. Raycroft, J. F., and Mastrangelo, M.: Massive intraperitoneal hemorrhage. A complication of disease of the gallbladder, Amer. J. Surg., 99:361, 1960.

6. Stahl, W. M., Jr.: Gastrointestinal-tract hemorrhage due to gallbladder disease, New Engl. J. Med., 260:471, 1959.